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VASCULAR DISEASE

OSTEOGENIC TRANSDIFFERENTIATION OF VASCULAR SMOOTH MUSCLE CELLS TO CALCIFYING VASCULAR CELLS IN 3D CULTURE: ENHANCEMENT BY LYSO-PHOSPHATIDYLCHOLINE AND ATTENUATION BY SCHNURRI-3

ACC Poster Contributions
Ernest N. Morial Convention Center, Hall F
Monday, April 04, 2011, 3:30 p.m.-4:45 p.m.

Session Title: Progenitor Cells, Endothelial Cells and Vascular Disease
Abstract Category: 8. Vascular Biology/Atherosclerosis/Thrombosis/Endothelium
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Authors: *Fernando Castro-Chavez, Joel D. Morrisett, Baylor College of Medicine, Houston, TX*

Background: We demonstrate the attenuation of human arteriosclerosis through a Runx2 inhibitor: Schnurri-3 (Shn3) using a 3D model of calcification in vitro of vascular smooth muscle cells (VSMCs) expressing an osteogenic phenotype that produces hydroxyapatite structures.

Methods: VSMCs were incubated with magnetite to obtain magnetically levitated 3D cultures with faster proliferation than conventional 2D cultures. VSMCs were grown either in normal DMEM, in DMEM plus osteogenic lyso-phosphatidylcholine (LPC), or in DMEM containing LPC plus Shn3 to attenuate calcification. Total protein, calcium, phosphate and glycosaminoglycans (GAGs) were determined, autofluorescence of organic hydroxyapatite was detected and images were acquired through an inverted microscope.

Results: When LPC was included in the media, the cell clusters exhibited translucent extensions and the cell pellets in 3D were enriched in total protein concentrations (see figure), calcium, phosphate and GAGs.

When excited, these extensions emitted intrinsic fluorescence, yet if treated with a FITC-conjugated fluorescent probe specific for hydroxyapatite, they emitted at the FITC wavelength. However, when Shn3 was added to the 3D culture containing LPC, the extensions were smaller or absent.

Conclusions: We present the 3D calcified structures formed by the VSMCs, and evidence for the attenuation of arteriosclerosis via Shn3, suggesting that similar structures are also present in vivo and that their attenuation is possible.